The complexities of heel ulcers

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Summary

This article examines the complexities of heel ulcers. Treatments now considered outdated and potentially hazardous are discussed as are modern treatments that may reduce the development of heel ulcers.

The incidence and prevalence of heel ulcers appear to be increasing (Collier 2000, Meehan 1994). This increase may suggest that practitioners have a low understanding of the causative factors or know the cause but do not always apply their knowledge to practice (Branemark et al 1996). The causes of heel ulceration are varied and complex (Box 1) and cannot simply be attributed to poor nursing, such as failure to relieve pressure (Dickson 1987) or inadequate pressure-relieving equipment.

Pressure and heel damage

Pressure is the force applied vertically to a surface. Shear is the force that is applied tangentially or in parallel (Bliss 1993) and is the term generally used when the force is acting uniformly in all directions.

Arteries divide into capillaries, which then form a network in subcutaneous tissues, dividing and sub-dividing as arterioles, forming capillaries, through to venules. This system is collectively known as the microcirculation, which supplies the tissues with nutrients and performs metabolite exchange and finally feeds into minor and then major veins. Blood pressure in the arterioles is provided by the heart and influenced by the release of vasodilator substances. Kosjak (1961) reported that microscopic examination of rat muscle 24 hours after being subjected to pressure of 70 mmHg for two hours showed a decrease or loss of cross-striations and myofilaments, hylaation of fibres and neutrophilic infiltrations. When complete relief of pressure was provided at regular five-minute intervals, as with an alternating pressure support system, the tissue showed consistently less change or no change at all when compared with tissues subjected to an equivalent amount of constant pressure. This was true even at pressures as high as 240 mmHg for three hours.

When pressure remains unrelieved, the tissues become inflamed and micro-thrombi form in the capillaries. If pressure continues, local ischaemia will lead to necrosis of the surrounding tissues. However, if the ischaemic reaction is interrupted before total obstruction of the tissues, reactive hyperaemia will occur and this reaction is proportional to the duration of the occlusion.

Bliss (1998) states: ‘Reactive hyperaemia is the vital process by which the body increases the blood flow to tissue which has been deprived of oxygen to restore levels to normal as quickly as possible.’ This suggests that the tissues ‘overreact’ to the occlusion and are flushed red with blood when vasodilation occurs. This action was also identified by Mayrovitz et al (1999) who suggest that a localised pressure-related tissue trauma is compensated for by substantial reperfusion. Metabolite removal and oxygen supply will then be quickly replenished providing the micro-thrombi and metabolites have not further occluded the vessels and further disrupted the supply. There are, however, those who doubt this explanation of reactive hyperaemia and believe that the presence of metabolites continues to degrade the tissues (Larcombe-McDouall et al 1998, McCord 1985). Interactions between the increased blood flow and metabolic activity at the ulcer edge might be oxygen-consuming, leading to reduced oxygen content passing through the capillaries and contributing to tissue ischaemia. Schubert (2000) found that interactions between the increased blood flow and metabolic activity at the ulcer edge might be oxygen-consuming, leading to reduced oxygen content passing through the capillaries and contributing to tissue ischaemia.

Increased pressure increases the risk of pressure damage and practitioners should be aware of the importance of relieving pressure. Pressure over the calcaneum can be three to five times greater over the bone than at the surface of the tissues (Le et al 1984). This effect is known as the cone of pressure (Figure 1) (David 1987). The depth of the pressure ulcer can be examined with a probe such as a swab (Figure 2).

A study by Mayrovitz et al (1999) found that hyperaemic responses were greatest at the pressure centre and diminished radially. It is important to understand this point as it helps to identify ulcers that are caused by direct pressure. These are sometimes anecdotally known as ‘inside out’...
pressure ulcers as the damage begins at the point of highest pressure (at the bone surface) and gradually advances to the lowest point of pressure (the surface). Non-blanching hyperaemia at the skin surface could be a sign that ischaemia and hidden damage are occurring deep in the tissues. In the author’s clinical experience, black or necrotic tissue rarely appears within hours of trauma. It can take up to several days for this damage to become noticeable, although a warning redness may be apparent in the first few hours. Ulcers that result from direct pressure are likely to be associated with the shape of the bone that is causing the pressure, which is often perfectly round and symmetrical in shape (Figure 2) (Hampton and Collins 2003).

Heel ulcers are potentially associated with sitting out of bed with heels on the floor (Collins 2000). As the feet take 19 per cent of the body weight, when heels are the only part of the feet in contact with the floor, the small heel area will be taking the full 19 per cent of weight (Collins 2000). In addition, one leg may be crossed over the other, thereby increasing the weight on the heel. The author has been asked on many occasions to suggest a more appropriate mattress as the condition of the patient’s heel pressure ulcer is either worsening or remaining static, only to find that the patient was sitting in a chair with the heel pressing on either the floor or a stool. The nurses appeared to be unaware that the floor or stool was harder than the mattress and likely to increase the risk of pressure ulcer development.

The duration of pressure is potentially significant in the formation of heel ulcers (Kosiak 1961). Even light loads will cause tissue ischaemia if unrelied for long periods of time (Kosiak 1961), if there is restricted flow and the capillary lining is distorted.

There are many devices and mattresses that prevent tissue damage (due to direct pressure) and, following individual patient assessment, these should be introduced when appropriate.

Shear
Shear is the stress resulting when one body attempts to slide past another and encounters resistance (Phillip 2003). It has already been noted

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**Box 1. Potential causes of heel ulcers**

- Direct pressure over the bony prominence: the heel has a relatively low resting blood perfusion level and is at increased risk of pressure damage (Donnelly 2001), particularly as the calcaneum has a small surface area and is a large weight-bearing bony prominence, subjecting the heel to high pressures (Donnelly 2001).
- Pressure and shear.
- Friction (Dinsdale 1974, Donnelly 2001).
- Arterial disease when the blood supply to the heels is poor.
- Anti-embolism stockings: Callam (1987) found 38 cases of damage caused through inappropriate use of anti-embolism stockings. Anti-embolism stockings may also be responsible for some pressure ulcers (Hannon 1977), particularly in patients with reduced circulation.
- Diabetes: increases the risk of pressure damage, particularly in the feet as ill-fitting shoes can cause pressure damage and poor circulation adds to the risk (Błaszczzyk et al 1998).
- Sedatives: the patient moves less frequently because of the sedation and this increases the risk of pressure damage.
- Drug therapy (for example, inotropes).
- Morbidity (Livesley and Chow 2002).
- Multiple sclerosis (Schneitzler 1978).
- Paraplegia (Meiners et al 2001).
- Epidural: Hughes (2001) found pressure damage following epidural in new mothers.
- Surgery: when blood pressure or local blood delivery is low.
- Poor perfusion: due to low resting perfusion level (Donnelly 2001) and critical skin ischaemia (cold; Raynaud's disease; chronic obstructive pulmonary disease; chronic cardiac failure; hypotension) (Donnelly 2001, Mathoko 1994, Schubert 1992).
- Neuropathy: when the patient is unable to identify pressure and pain and is unaware that he or she should change position.
- Heavy bed covers: these may press onto the toes and push the heel harder onto the bed surface. This additional pressure could potentially increase the risk of pressure damage.
- Poor seating posture: this could be caused by incorrect chair size for the patient (Collins 2000).
- Use of inappropriate equipment: such as water-filled gloves (Donnelly 2001, Lockyer-Stevens 1993, Williams 1993).

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**Figure 1. The cone of pressure (David 1987)**

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**Figure 2. Undermining identified with a probe such as a swab (undermining level demonstrated by curved line)**

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that shear is force that is applied tangentially or in parallel (Bliss 1993). This can best be demonstrated by placing the knuckle of the finger in the palm of the hand and rocking the knuckle back and forth. The tissue will remain in one place and the bone will move over the inside of the tissues. As the bone makes this movement, it will wrinkle and pinch the loose tissue and this has the same effect on the capillaries as if a straw has been bent. Trying to suck through a bent straw is impossible; likewise, blood will not pass through a deformed capillary and the tissues will be starved of oxygen and nutrients. Bennett et al (1974) demonstrated that the pressure level capable of disrupting blood flow can be reduced by half in the presence of shear forces. This means that shear forces and pressure are powerful antagonists of tissue.

The damage caused by shearing forces is different to that caused by direct pressure (Figures 3 and 4). The damage can anecdotally be described as an ‘outside in’ injury and manifests itself as an uneven and more shallow injury than that resulting from pure pressure. The two types can be found together forming a deep and uneven ulcer. Shearing force pressure ulcers are potentially associated with sliding down the bed or not supporting the heel when it is on the floor (when the patient is sitting in a chair). The solutions are simple. The patient should be provided with the correct size chair (Collins 1998a, 1998b, 2000) which will place the feet at a 90° angle to the legs, that is flat on the floor. Pressure will then be removed from the bony prominence.

When in bed, the patient should be supported either with an electric profiling bed with the break at the knee (so that the knee bends over the profiled area) or with a mattress that prevents the patient from slipping down in the bed (Hampton 1998).

Friction
Friction is caused by the rubbing together of two surfaces, which can occur when the skin is rubbed against shoes, sheets or the floor either through natural movement of the patient or through poor lifting and handling techniques that do not ensure that the patient is lifted clear of the underlying surface (Dinsdale 1974).

The skin has a stratified, impermeable and avascular layer of dead keratinised skin scales (epidermis), which is waterproof, prevents water loss, and acts as a barrier against bacterial invasion of the dermis. Healthy skin has a top layer of stratum corneum (horny layer), which consists of dead cells that have been propelled away from the base of the epidermis by layers of cells growing below in the dermis. The flattened dead cells are grouped around lipids and proteins and this mixture is the skin’s main barrier to water loss. Any break in this layer can pose a threat to the systems of the body.

The epidermis invaginates the surface, forming hair follicles and exocrine glands. Underlying the epidermis are the viable dermis and subcutaneous layers consisting of nerves, hair follicles, sweat and sebaceous glands and adipose (fatty) tissue. These structures are maintained and supported by fibrous and elastic connective tissue with each structure having a special role to play in maintaining homeostasis. The surface epidermis and dermis are held together firmly by interlinking fingerlike projections (Green 1991).

When friction occurs, it places immense strain on the projections between the epidermis and dermis and this can separate the two layers. When this occurs, fluid floods the space that is left following the separation, which lifts the epidermis into a blister. If the blister is deroofed, the dermis is exposed and as this is the layer with nerve endings, the area becomes painful (Flanagan 1997). The blister can be left to be naturally absorbed by the body or sometimes it is deroofed to release the underlying fluid. If the latter option is selected, then the wound requires extra care as it is open to infection. In the author’s opinion it is wiser to cover the blister with either film or thin hydrocolloid and leave it until the body has absorbed the fluid and the blister naturally de-roofs. Some blisters can turn black as the surface tissue dies. These blisters can become hard and appear similar to a necrotic area that has formed due to direct pressure. There are, however, subtle differences, which are obvious to the experienced eye.
A pressure ulcer that results from direct pressure is often dark red in the surrounding tissues where pressure damage continues under the surface. A black blister is often moveable with the fingers or feels shallow to touch. If the area can be identified as a blackened blister, it is probably best left dry and will drop off fairly quickly. However, if the inexperienced of the assessor means identifying the depth of tissue damage is not possible – for example, if the patient is in a nursing home – then it would be wise to treat for the more serious condition. This would lead to debridement of the necrotic area by first applying moist dressings to rehydrate the tissue (providing there is a good vascular supply and a potential for healing). Once rehydration is complete, the tissue can be debrided with iodine cadexomers, larval therapy, hydrogels or hydrocolloids. Sharp and surgical debridement are faster options, but nurses should only undertake sharp debridement when competent to do so. If the wound is necrotic, there is a high potential for clinical infection and, therefore, the tissue must be removed – regardless of the healing potential or the vascular status (Fowler and van Rijswijk 1995). The safest method of tissue removal is autolysis, which can be initiated by application of moist dressings or larval therapy (Atiyeh et al 2002, Sherman et al 2001, Sibbald et al 2000).

Heel protectors in the form of devices and dressings should be used to protect and prevent deterioration of the tissues. Hoists or sliding sheets should be used when moving patients to prevent shearing forces on the skin during lifting.

Diabetes
At any one time, there are more patients occupying hospital beds with diabetic foot complications than with all other complications of diabetes, and 50 per cent of all non-traumatic amputations are performed on patients with diabetes (Barnett 1992). Sensory neuropathy or damage to the sensory nerves in legs and feet leading to loss of sensation and potential injury that goes unnoticed can lead to necrosis. Motor neuropathy creates a wasting of muscles in the feet, causing deformities, and pressure damage from shoes also increases the potential risk of developing heel ulcers.

In atherosclerosis, monocytes adhere to the lining of the arterial wall, encouraging macrophages to release growth factors, stimulating growth and migration of vascular smooth muscle cells. Platelets then adhere resulting in a plaque that narrows or blocks the lumen of the artery (Bryant 1992). In diabetes, this process is accelerated. Arterial calcification is found in 94 per cent of patients who have had diabetes for 35 years (Laing 1998). In addition, non-enzymatic glycation of soft tissue in diabetes means that skin and soft tissue biomechanics are altered and the skin is stiffer than normal (Hashmi 2000, Jude and Boulton 1998). Non-enzymatic glycation is the mechanism by which proteins, such as collagen, are subject to chronic attack from glucose (Majno and Joris 1996).

All these abnormalities place the patient with diabetes at greater risk from pressure and shear damage. The diabetic heel ulcer is a significant healthcare problem and inadequate or improper therapy may lead to amputation. Therefore, careful monitoring and patient and healthcare professional education are imperative.

Arterial disease
Arterial ulcers can be divided into two main groups of macro- and micro-vascular disease; both types will affect the formation of heel ulcers. Other types that form under these headings are the neuro-ischaemic or autoimmune disorders such as vasculitis found in rheumatoid arthritis.

The signs and symptoms of macro-vascular arterial disease (Figure 6) are varied but there are often consistencies that would lead the assessor to conclude that arterial disease may be present. There may be reports of pain at night and/or pain on walking such that the patient has to stop after a few minutes as the pain is relieved by rest. The foot is quite often cold to touch and may be numb. There may be skin pallor and the leg may be hairless and shiny, with smooth, thin,
tautly stretched skin. Pinching the nail bed shows the sluggishness of refill time (longer than two seconds). Lifting the leg higher than the heart produces a cadaveric pallor and lowering the leg shows a delay in returning to normal colour with a possible final dependent dark red discoloration (rubor). In addition to the tissue damage, the leg and toes may be very pale. These symptoms alone should alert any assessor to the potential of developing heel ulcers.

Anti-embolism stockings
Anti-embolism stockings should not be used for patients with arterial disease, diabetes or rheumatoid arthritis without Doppler assessment as the damage to poorly perfused heels and feet (Figures 7 and 8) can be destructive. However, clinical risk of developing deep vein thrombosis and pulmonary embolism needs to be assessed and decisions made about alternative prophylaxis.

The patient in Figures 7 and 8 was paraplegic, diabetic and had arterial disease. He was prescribed anti-embolism stockings during surgery and although it is unclear whether the consequent tissue damage was due to the stockings or to pressure during surgery, he died when his pressure ulcer caused a systemic reaction (sepsicaemia).

The relationship of other factors to formation of heel ulcers
Any type of drug that causes sedation or anaesthesia will reduce frequency of movement by the patient and thereby increase the risk of pressure ulcer formation on the heels. Decreased blood pressure during surgery or following shock will lower blood delivery to the peripheries, such as the heels, and this leads to an increased risk of tissue damage (Donnelly 2001, Hampton 1997).

Prevention of heel ulcers and recommended practice
Mattresses do not always protect the heel adequately, for the reasons outlined above. Pillows are often used to support the leg, leaving the heel free of pressure as it hangs over the edge of the pillow. However, the pillow (or the patient) can move and the heel can end up directly on the pillow with a potential increase in pressure over the bony prominence. There is also a possibility that strain will occur over the Achilles tendon placing that area at increased risk. The nurse should assess the risk for each patient and make a decision on whether pillows should be used for protection of the heel. This decision should take into account the potential for the development of a pressure ulcer in both scenarios, when the pillow is or is not used.

In the past, two hourly turns were the key tenet of pressure ulcer prevention (Bell et al 1974), although there has been little evidence to support the overall efficacy (Donnelly 2001). Assessment and planning for the protection of heels should be different from normal assessment as the heels will not respond under pressure in the same manner as the sacral area.

Bull (1930) recommended rubbing pressure areas with a little cotton wool soaked in skin stimulant, such as eau de cologne or methylated spirit, and this method became, for many years, the favoured prevention of pressure ulcers. Dyson (1978) compared two groups of patients: those who received massage of pressure areas as part of their preventive treatment and those who did not receive massage. Post-mortem examinations showed a difference between the two groups with the non-massaged group having a lower incidence of pressure ulcers and the massaged group showing macerated and degenerated tissues in the areas that were massaged. The author suggests there is a possibility that this is the result of drawing blood to the surface tissues through the massage action, leaving the area next to the bone deficient in blood.

Water-filled gloves were popular in some areas but are not now recommended as the pressures achieved with these cannot be judged correctly and could lead to increased damage (Donnelly 2001, Lockyer-Stevens 1993, NICE 2001, Williams 1993). The author has heard (anecdotally) of the practice of filling a lemonade bottle with water and placing it under the Achilles tendon. This is an unproven method of treatment and is dangerous as the heel could slip and come in contact with the bottle, thereby greatly increasing pressure over the bony prominence. This practice should be discouraged.

Total relief of pressure is difficult to achieve. However, there are several heel appliances marketed for shear, friction and pressure. Heel booties are available, made of air-filled static cells with...
no cells over the heel, sustaining a pressure-free zone in that area. There are also bootees consisting of a soft outer cover with a gel-filled zone. These bootees will support the foot and Achilles tendon, while reducing pressure under the heel.

**Conclusion**

The complexities of pressure ulcer formation on the heel can be assessed, and, using an evidence-based approach, these ulcers can generally be avoided. The use of pillows, massage and water-filled gloves under the ankles should be discontinued unless the practitioner can be assured that the heel will remain free from contact with these items and associated pressure. The united forces of pressure, shear and friction are powerful and require proactive and clinically sound measures to prevent damage occurring. Protecting the heels against skin damage is part of high quality care. The use of pressure relieving and reducing equipment is extremely important but it is only one component of the overall requirements of protection. The use of bed cradles helps to relieve some of the pressure and the use of hoists and slide sheets under the heels (as well as the sacrum) will reduce the potential for skin damage caused by inappropriate lifting techniques.

Correct positioning in a chair that is an appropriate size for the individual will redirect the pressure from the heel and lessen the potential for pressure damage. Nurses should be aware that patients with diabetes and neuropathy, paraplegia, arterial disease or multiple sclerosis, and those patients taking certain drugs such as isotropes or sedatives, will have increased potential for pressure damage and therefore increased protection should be provided for the heels in the form of pressure relieving cushions or mattresses under the feet.

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